* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page for STN Seminar Schedule - N. America
NEWS 2	OCT 04	Precision of EMBASE searching enhanced with new chemical name field
NEWS 3	OCT 06	Increase your retrieval consistency with new formats or for Taiwanese application numbers in CA/CAplus.
NEWS 4	OCT 21	CA/CAplus kind code changes for Chinese patents increase consistency, save time
NEWS 5	OCT 22	New version of STN Viewer preserves custom highlighting of terms when patent documents are saved in .rtf format
NEWS 6	OCT 28	INPADOCDB/INPAFAMDB: Enhancements to the US national patent classification.
NEWS 7	NOV 03	New format for Korean patent application numbers in CA/CAplus increases consistency, saves time.
NEWS 8	NOV 04	Selected STN databases scheduled for removal on December 31, 2010
NEWS 9	NOV 18	PROUSDDR and SYNTHLINE Scheduled for Removal December 31, 2010 by Request of Prous Science
NEWS 10	NOV 22	Higher System Limits Increase the Power of STN Substance-Based Searching
NEWS 11	NOV 24	Search an additional 46,850 records with MEDLINE backfile extension to 1946
NEWS 12	DEC 14	New PNK Field Allows More Precise Crossover among STN Patent Databases
NEWS 13	DEC 18	ReaxysFile available on STN
NEWS 14	DEC 21	CAS Learning Solutions -- a new online training experience
NEWS 15	DEC 22	Value-Added Indexing Improves Access to World Traditional Medicine Patents in CAplus
NEWS 16	JAN 24	The new and enhanced DPCI file on STN has been released
NEWS 17	JAN 26	Improved Timeliness of CAS Indexing Adds Value to USPATFULL and USPAT2 Chemistry Patents
NEWS 18	JAN 26	Updated MeSH vocabulary, new structured abstracts, and other enhancements improve searching in STN reload of MEDLINE
NEWS 19	JAN 28	CABA will be updated weekly
NEWS 20	FEB 23	PCTFULL file on STN completely reloaded
NEWS 21	FEB 23	STN AnaVist Test Projects Now Available for Qualified Customers
NEWS 22	FEB 25	LPCI will be replaced by LDPCI
NEWS 23	MAR 07	Pricing for SELECTing Patent, Application, and Priority Numbers in the USPAT and IFI Database Families is Now Consistent with Similar Patent Databases on STN

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FILE 'HOME' ENTERED AT 20:28:24 ON 30 MAR 2011

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FILE 'CAPLUS' ENTERED AT 20:28:36 ON 30 MAR 2011
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=> antigen (s) (TRAIL-R3)
L1             6 ANTIGEN (S) (TRAIL-R3)

=> antigen (s) Sp1
L2           254 ANTIGEN (S) SP1

=> gene (w) therapy
L3        107698 GENE (W) THERAPY

=> DNA (w) vaccine
L4         13045 DNA (W) VACCINE

=> antigen (s) L4
L5          3109 ANTIGEN (S) L4

=> (TRAIL-R) and L5
L6             0 (TRAIL-R) AND L5

=> OX40 and L5
L7             3 OX40 AND L5

=> (Ap-1) and L4
L8             4 (AP-1) AND L4

=> RANK and L3
AND IS NOT VALID HERE
The term is either unrecognized or invalid.

=> L3 and RANK
L9           114 L3 AND RANK

=> antigen (s) L9
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'ANTIGEN (S) L25'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'ANTIGEN (S) L26'
L10           18 ANTIGEN (S) L9

=> D L1 IBIIB AES 1-6
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individual files.
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L1 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2009:1432065 CAPLUS
DOCUMENT NUMBER: 152:284029
TITLE: Prognostic significance of tumour necrosis factor-related apoptosis-inducing ligand (TRAIL) receptor expression in patients with breast cancer
AUTHOR(S): Ganten, Tom M.; Sykora, Jaromir; Koschny, Ronald; Batke, Emanuela; Aulmann, Sebastian; Mansmann, Ulrich; Stremmel, Wolfgang; Sinn, Hans-Peter; Walczak, Henning
CORPORATE SOURCE: Division of Apoptosis Regulation (D040), German Cancer Research Center (DKFZ), Heidelberg, Germany
SOURCE: Journal of Molecular Medicine (Heidelberg, Germany) (2009), 87(10), 995-1007
CODEN: JMLME8; ISSN: 0946-2716
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English
OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2008:288351 CAPLUS
DOCUMENT NUMBER: 149:375256
TITLE: Tumor necrosis factor-related apoptosis inducing ligand-R4 decoy receptor expression is correlated with high Gleason scores, prostate-specific antigen recurrence, and decreased survival in patients with prostate carcinoma
AUTHOR(S): Koksal, Ismail T.; Sanlioglu, Ahter D.; Karacay, Bahri; Griffith, Thomas S.; Sanlioglu, Salih
CORPORATE SOURCE: Human Gene Therapy Unit and the Department of Medical Biology and Genetics, Faculty of Medicine, Akdeniz University, Antalya, Turk.
SOURCE: Urologic Oncology: Seminars and Original Investigations (2008), 26(2), 158-165
CODEN: UOSOAA; ISSN: 1078-1439
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:1023171 CAPLUS
DOCUMENT NUMBER: 147:371785
TITLE: Engineered antibody drug conjugates with defined sites

and stoichiometries of drug attachment having cytotoxic activity against antigen-specific targets
INVENTOR(S): McDonagh, Charlotte; Carter, Paul
PATENT ASSIGNEE(S): Seattle Genetics, Inc., USA
SOURCE: PCT Int. Appl., 149pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007103288	A2	20070913	WO 2007-US5552	20070302
WO 2007103288	A3	20071129		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRIORITY APPLN. INFO.:	US 2006-778472P	P	20060302
	US 2006-872348P	P	20061201

OTHER SOURCE(S): MARPAT 147:371785

L1 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004112706	A2	20041229	WO 2004-US19028	20040614
WO 2004112706	A3	20050414		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,

EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2004249191	A1	20041229	AU 2004-249191	20040614
AU 2004249191	B2	20110106		
CA 2529051	A1	20041229	CA 2004-2529051	20040614
EP 1633372	A2	20060315	EP 2004-755303	20040614
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
JP 2007502868	T	20070215	JP 2006-533794	20040614
US 20070104686	A1	20070510	US 2004-560653	20040614
PRIORITY APPLN. INFO.:			US 2003-478187P	P 20030613
			US 2003-478230P	P 20030613
			US 2003-478250P	P 20030613
			WO 2004-US19028	W 20040614

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

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REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2000:583433 CAPLUS
DOCUMENT NUMBER: 134:146230
TITLE: Expression of TRAIL receptors in human autoreactive and foreign antigen-specific T cells
AUTHOR(S): Wendling, U.; Walczak, H.; Dorr, J.; Jaboci, C.; Weller, M.; Krammer, P. H.; Zipp, F.
CORPORATE SOURCE: Division of Neuroimmunology, Department of Neurology, Charite, Berlin, Germany
SOURCE: Cell Death and Differentiation (2000), 7(7), 637-644
CODEN: CDDIEK; ISSN: 1350-9047
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
OS.CITING REF COUNT: 40 THERE ARE 40 CAPLUS RECORDS THAT CITE THIS RECORD (41 CITINGS)
REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 6 OF 6 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | Citing References

ACCESSION NUMBER: 2000:399144 BIOSIS
DOCUMENT NUMBER: PREV200000399144
TITLE: Expression of TRAIL receptors in human autoreactive and foreign antigen-specific T cells.
AUTHOR(S): Wendling, U.; Walczak, H.; Doerr, J.; Jaboci, C.; Weller, M.; Krammer, P. H.; Zipp, F. [Reprint author]
CORPORATE SOURCE: Department of Neurology, Division of Neuroimmunology, University Hospital Charite, Augustenburger Platz 1, Campus Virchow, Forschungshaus, 2.OG, R. 535, 13353, Berlin, Germany
SOURCE: Cell Death and Differentiation, (July, 2000) Vol. 7, No. 7, pp. 637-644. print.
ISSN: 1350-9047.
DOCUMENT TYPE: Article
LANGUAGE: English

ENTRY DATE: Entered STN: 20 Sep 2000
Last Updated on STN: 8 Jan 2002

=> D L7 IBIB ABS 1-3

L7 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:859817 CAPLUS
DOCUMENT NUMBER: 147:298670
TITLE: Enhanced protective efficacy and reduced viral load of foot-and-mouth disease DNA vaccine with co-stimulatory molecules as the molecular adjuvants
AUTHOR(S): Xiao, Chong; Jin, Huali; Hu, Yanxin; Kang, Youmin; Wang, Junpeng; Du, Xiaogang; Yang, Yu; She, Ruiping; Wang, Bin
CORPORATE SOURCE: State Key Laboratory for Agro-Biotechnology, Key Laboratory of Agro-Microbial Resources and Applications of MOA, China Agricultural University, Beijing, 100094, Peop. Rep. China
SOURCE: Antiviral Research (2007), 76(1), 11-20
CODEN: ARSRDR; ISSN: 0166-3542
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To improve efficacy of DNA vaccination, various approaches have been developed, including the use of plasmid expressing co-stimulatory mols. as mol. adjuvants. Here, the authors investigated whether co-inoculation of a construct expressing either 4-1BBL or OX40L as the mol. adjuvant with FMDV DNA vaccine, pcD-VP1, can increase immune responses and protective efficacies. Compared to the group immunized with pcD-VP1 alone, the co-inoculation of either mol. adjuvant induced a higher ratio of IgG2a/IgG1, higher levels of expression of IFN-γ in CD4+ and CD8+ T cells and antigen-specific CTL responses, and more importantly provided an enhanced protection against the live FMDV challenge in animals. Concurrently, 4-1BBL as the mol. adjuvant dramatically reduced the viral loads of FMDV in vivo after the challenge. Thus, co-stimulatory mols. 4-1BBL and OX40L can enhance the **antigen**-specific cell-mediated responses elicited by VP1 **DNA vaccine** and provide an enhanced protective efficacy with the reduced viral loads.

OS.CITING REF COUNT: 9 THERE ARE 9 CAPLUS RECORDS THAT CITE THIS RECORD (9 CITINGS)
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: **DNA vaccines** comprising immunomodulatory proteins and **antigen** from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004112706	A2	20041229	WO 2004-US19028	20040614
WO 2004112706	A3	20050414		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004249191	A1	20041229	AU 2004-249191	20040614
AU 2004249191	B2	20110106		
CA 2529051	A1	20041229	CA 2004-2529051	20040614
EP 1633372	A2	20060315	EP 2004-755303	20040614
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
JP 2007502868	T	20070215	JP 2006-533794	20040614
US 20070104686	A1	20070510	US 2004-560653	20040614
PRIORITY APPLN. INFO.:			US 2003-478187P	P 20030613
			US 2003-478230P	P 20030613
			US 2003-478250P	P 20030613
			WO 2004-US19028	W 20040614

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, **Ox40**, **Ox40** ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1998:684978 CAPLUS
DOCUMENT NUMBER: 129:274700
ORIGINAL REFERENCE NO.: 129:56017a,56020a
TITLE: DNA encoding targeting protein fused to **antigen** or epitope in enhancement of immune response to **DNA vaccines**
INVENTOR(S): Boyle, Jefferey Stephen; Brady, Jamie Louise; Lew, Andrew Mark
PATENT ASSIGNEE(S): The Council of the Queensland Institute of Medical Research, Australia; Commonwealth Scientific and Industrial Research Organisation; The University of Melbourne; The Walter and Eliza Hall Institute of

Medical Research; CSL Ltd.
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9844129	A1	19981008	WO 1998-AU208	19980326
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2285692	A1	19981008	CA 1998-2285692	19980326
AU 9864902	A	19981022	AU 1998-64902	19980326
AU 728962	B2	20010125		
EP 972054	A1	20000119	EP 1998-910530	19980326
EP 972054	B1	20081210		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
NZ 500151	A	20010126	NZ 1998-500151	19980326
JP 2001522235	T	20011113	JP 1998-540989	19980326
JP 4382163	B2	20091209		
AT 417112	T	20081215	AT 1998-910530	19980326
ZA 9802608	A	19981008	ZA 1998-2608	19980327
US 20030035793	A1	20030220	US 2002-185318	20020628
US 7423016	B2	20080909		
US 20030072742	A1	20030417	US 2002-185799	20020628
US 7423023	B2	20080909		
CA 2489940	A1	20060608	CA 2004-2489940	20041208

PRIORITY APPLN. INFO.:

AU 1997-5891	A	19970327
AU 1998-1830	A	19980213
WO 1998-AU208	W	19980326
US 2000-402020	A1	20000328

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The present invention provides methods of enhancing the immune response to an immunogen and to compns. for use in these methods. In particular the present invention provides a DNA mol. for use in raising an immune response to an antigen. The DNA mol. includes a first sequence encoding a targeting mol., a second sequence encoding the antigen or an epitope thereof, and optionally a third sequence encoding a polypeptide which promotes dimerization or multimerization of the product encoded by the DNA mol. Immunization of mice with a no. of DNA sequences encoding CTLA4-antigen fusions enhanced the immune response to the antigen.

OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D L8 IBIB ABS 1-4

L8 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:284018 CAPLUS
DOCUMENT NUMBER: 146:289496
TITLE: Human herpesvirus-derived promoters for introducing gene into lymphocyte and application thereof
INVENTOR(S): Takemoto, Masaya; Mori, Yasuko; Yamanishi, Koichi; Fuke, Isao; Gomi, Yasuyuki; Takahashi, Michiaki
PATENT ASSIGNEE(S): The Research Foundation for Microbial Diseases of Osaka University, Japan
SOURCE: PCT Int. Appl., 119pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007029712	A1	20070315	WO 2006-JP317574	20060905
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2006288279	A1	20070315	AU 2006-288279	20060905
CA 2621917	A1	20070315	CA 2006-2621917	20060905
EP 1932911	A1	20080618	EP 2006-797474	20060905
R: BE, DE, FR, GB, IT, NL				
CN 101300350	A	20081105	CN 2006-80041278	20060905
CN 101906418	A	20101208	CN 2010-10226760	20060905
IN 2008CN01631	A	20081226	IN 2008-CN1631	20080401
KR 2008036244	A	20080425	KR 2008-7007967	20080402
US 20100005536	A1	20100107	US 2008-991637	20080716
US 20090208516	A1	20090820	US 2008-195647	20080821
US 20090214579	A1	20090827	US 2008-195665	20080821

PRIORITY APPLN. INFO.:

JP 2005-261366	A	20050908
CN 2006-80041278	A3	20060905
WO 2006-JP317574	W	20060905
US 2008-991637	A3	20080716

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB This invention relates to a promoter for inducing expression selectively and strongly in an immunocompetent cell and/or a blood cell such as a lymphocyte. It is based on a finding that HHV6 MIE promoter, HHV7 MIE promoter and HHV7 U95 promoter induce a specific expression in an immunocompetent cell and/or a blood cell such as a T lymphocyte. By utilizing the promoters, a selective delivery of a **DNA vaccine** or the like can be realized.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: **DNA vaccines** comprising immunomodulatory proteins and antigen from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004112706	A2	20041229	WO 2004-US19028	20040614
WO 2004112706	A3	20050414		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004249191	A1	20041229	AU 2004-249191	20040614
AU 2004249191	B2	20110106		
CA 2529051	A1	20041229	CA 2004-2529051	20040614
EP 1633372	A2	20060315	EP 2004-755303	20040614
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
JP 2007502868	T	20070215	JP 2006-533794	20040614
US 20070104686	A1	20070510	US 2004-560653	20040614
PRIORITY APPLN. INFO.:			US 2003-478187P	P 20030613
			US 2003-478230P	P 20030613
			US 2003-478250P	P 20030613
			WO 2004-US19028	W 20040614

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, **AP-1**, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, Ox40, Ox40 ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:794545 CAPLUS
DOCUMENT NUMBER: 141:289084
TITLE: Composition for inducing immunotolerance
INVENTOR(S): Van Oosterhout, Antonius Josephus Maria; Kapsenberg, Martien Lukas; Weller, Frank Reinoud; Taher, Yousef Al-Madane; Lobato-Van Esch, Elisabeth Catharina Adriana Maria; Vissers, Joost Lambert Max
PATENT ASSIGNEE(S): Universiteit Utrecht Holding B.V., Neth.
SOURCE: Eur. Pat. Appl., 23 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1462111	A1	20040929	EP 2003-75909	20030328
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CA 2518793	A1	20041007	CA 2004-2518793	20040325
WO 2004084927	A2	20041007	WO 2004-NL205	20040325
WO 2004084927	A3	20050127		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1608391	A2	20051228	EP 2004-723429	20040325
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				
EP 1772152	A2	20070411	EP 2006-77139	20040325
EP 1772152	A3	20070627		
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR				
EP 1842550	A2	20071010	EP 2007-105399	20040325
EP 1842550	A3	20081210		
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LI, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR				
US 20060057154	A1	20060316	US 2005-229333	20050915
PRIORITY APPLN. INFO.:			EP 2003-75909	A 20030328
			EP 2004-723429	A3 20040325
			WO 2004-NL205	W 20040325

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention provides methods of treating allergic disorders and compns. for use therein. The methods comprise administering an allergen and one or more medicaments. These medicaments are compds. that inhibit the transcription of genes involved in the initiation of innate and specific immunity, thereby promoting the development of tolerance to these allergens, through inhibition of the NF-κB and/or the MAPK/AP-1 signal transduction pathway(s). In another embodiment, the use of **DNA vaccines** is disclosed that incorporate a gene encoding one or more

allergen sequences or fragments thereof, in combination with genes encoding proteins that inhibit the activation of the NF-κB and/or the MAPK/**AP-1** pathway or in combination with small interfering RNA sequences or anti-sense sequences that inhibit the expression of NF-κB and/or **AP-1** proteins.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:946139 CAPLUS
DOCUMENT NUMBER: 138:38057
TITLE: Chimeric antigens and vectors for targeted delivery in DNA vaccination
INVENTOR(S): Valiante, Nicholas
PATENT ASSIGNEE(S): Chiron S.p.A., Italy; Chiron S.r.L.
SOURCE: PCT Int. Appl., 30 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002098456	A2	20021212	WO 2002-IB3105	20020530
WO 2002098456	A3	20040506		
W: US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
EP 1440156	A2	20040728	EP 2002-751532	20020530
EP 1440156	B1	20080827		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
AT 406449	T	20080915	AT 2002-751532	20020530
US 20040147721	A1	20040729	US 2003-479649	20031201
US 7541180	B2	20090602		
US 20100098718	A1	20100422	US 2009-455444	20090601

PRIORITY APPLN. INFO.:

GB 2001-13798	A	20010606
WO 2002-IB3105	W	20020530
US 2003-479649	A1	20031201

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The author discloses chimeric antigens comprising a dimer of first fusion protein with a second fusion protein. The fusion proteins comprise a targeting domain, a leucine zipper domain, and optionally an antigen for the second fusion protein.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D L18 IBIB ABS 1-18
L18 NOT FOUND
The L-number entered has not been defined in this session, or it has been deleted. To see the L-numbers currently defined in this session, enter DISPLAY HISTORY at an arrow prompt (=>).

=>
=> D L10 IBIB ABS 1-18

L10 ANSWER 1 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2009:1566750 CAPLUS
DOCUMENT NUMBER: 152:67621
TITLE: β-Adrenergic receptor agonists for the treatment of B-cell proliferative disorders
INVENTOR(S): Rickles, Richard; Lee, Margaret S.
PATENT ASSIGNEE(S): CombinatoRx, Inc., USA
SOURCE: PCT Int. Appl., 111 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2009151569	A2	20091217	WO 2009-US3449	20090608
WO 2009151569	A3	20100225		

W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

US 20100009934	A1	20100114	US 2009-480034	20090608
PRIORITY APPLN. INFO.:			US 2008-60064P P	20080609

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention discloses a method for treating a B-cell proliferative disorder by administering to a patient a β-Adrenergic receptor (BAR) agonist, e.g., formulated for administration by a route other than inhalation (such as for oral or i.v. administration), in an amt. effective to treat the B-cell proliferative disorder. The BAR agonist may be administered as a monotherapy or in combination with one or more other agents, e.g., a PDE inhibitor, an A2A receptor agonist, or an antiproliferative compd., in amts. that together are effective to treat the B-cell proliferative disorder. The invention further discloses pharmaceutical compns. and kits including a BAR agonist, alone or in combination with addnl. agents, for the treatment of a B-cell proliferative disorder.

L10 ANSWER 2 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2009:86451 CAPLUS
DOCUMENT NUMBER: 150:160095
TITLE: Use of adenosine A2A receptor agonists and phosphodiesterase (PDE) inhibitors for the treatment of B-cell proliferative disorders, and combinations

with other agents
INVENTOR(S): Rickles, Richard; Lee, Margaret S.
PATENT ASSIGNEE(S): CombinatoRx, Incorporated, USA
SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2009011893	A2	20090122	WO 2008-US8758	20080717
WO 2009011893	A3	20090319		
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA			
AU 2008276451	A1	20090122	AU 2008-276451	20080717
CA 2694983	A1	20090122	CA 2008-2694983	20080717
US 20090053168	A1	20090226	US 2008-175219	20080717
EP 2178369	A2	20100428	EP 2008-780231	20080717
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, AL, BA, MK, RS				

PRIORITY APPLN. INFO.:

US 2007-950307P	P	20070717
US 2007-965587P	P	20070821
WO 2008-US8758	W	20080717

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The invention provides compns. and methods for the treatment of B-cell proliferative disorders that employ an A2A receptor agonist or one or more PDE inhibitors. The methods and compns. may further include an antiproliferative compd.
OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

L10 ANSWER 3 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 2007:932900 CAPLUS
DOCUMENT NUMBER: 147:297111
TITLE: Polynucleotides and polypeptide sequences involved in the process of bone remodeling
INVENTOR(S): Sooknanan, Roy Rabindranauth; Tremblay, Gilles Bernard; Filion, Mario
PATENT ASSIGNEE(S): Alethia Biotherapeutics Inc., Can.
SOURCE: PCT Int. Appl., 203pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007093042	A1	20070823	WO 2007-CA210	20070213
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2007215334	A1	20070823	AU 2007-215334	20070213
CA 2638823	A1	20070823	CA 2007-2638823	20070213
EP 1994155	A1	20081126	EP 2007-710624	20070213
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
JP 2009525730	T	20090716	JP 2008-553592	20070213
US 20090298763	A1	20091203	US 2009-279054	20090113
US 20100104575	A1	20100429	US 2009-580943	20091016
PRIORITY APPLN. INFO.:			US 2006-772585P	P 20060213
			US 2006-816858P	P 20060628
			WO 2007-CA210	W 20070213
			US 2009-279054	A2 20090113

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB This invention relates, in part, to unique and newly identified genetic polynucleotides involved in the process of bone remodeling, variants and derivs. of the polynucleotides and corresponding polypeptides, uses of the polynucleotides, polypeptides, variants and derivs., and methods and compns. for the amelioration of symptoms caused by bone remodeling disorders. Human polynucleotides were identified using macroarrays prepd. using RAMP amplified RNA from human precursor cells, differentiated intermediate and mature osteoclasts for four human donors, and 30 different normal human tissues. The RAW 264.7 osteoclast precursor cell line and human precursor cells (peripheral blood mononuclear cells or CD34-pos. progenitors) are well known in the art as murine and human models of osteoclastogenesis; human primary osteoclasts were differentiated from G-CSF-mobilized peripheral blood mononuclear cells in the presence of M-CSF and **RANK** ligand. Identification and validation of the polynucleotides involved in osteoclast activity confirms their potential as therapeutic targets and use uses for the amelioration of disease states and research purposes.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 4 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:770027 CAPLUS
DOCUMENT NUMBER: 147:141447
TITLE: Canine receptor activator of NF-κB ligand and methods for its preparation and use in treating conditions associated with loss of bone minerals
INVENTOR(S): Mattson, Jeanine D.; McClanahan, Terrill

PATENT ASSIGNEE(S): Schering-Plough Ltd., Switz.
SOURCE: PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004052233	A2	20040624	WO 2003-US39292	20031210
WO 2004052233	A3	20071206		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MA, MD, MG, MK, MN, MX, MZ, NI, NO, NZ, PG, PH, PL, PT, RO, RU, SC, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, US, UZ, VC, VN, YU, ZA, ZM				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, AP, EA, EP, OA				
CA 2508773	A1	20040624	CA 2003-2508773	20031210
JP 2006521084	T	20060921	JP 2004-558667	20031210
US 20060154858	A1	20060713	US 2005-537864	20050607
US 7462700	B2	20081209		
US 20090148456	A1	20090611	US 2008-266359	20081106
JP 2010042036	A	20100225	JP 2009-268156	20091125

PRIORITY APPLN. INFO.:			
US 2002-432092P	P	20021210	
JP 2004-558667	A3	20031210	
WO 2003-US39292	W	20031210	
US 2005-537864	A3	20050607	

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Nucleic acid mols. that encode a substantial part of canine receptor activator of NF-κB ligand (RANKL) polypeptide are isolated from a canine splenocyte cDNA library, including the extracellular domains of the polypeptide, the full-length polypeptide, and fragments thereof. Vectors and host cells encoding and expressing canine RANKL polypeptide are provided, as well as rat monoclonal antibodies that bind to RANKL and that inhibit RANKL activity. Canine **RANK** may be used in methods of treating an animal to inhibit or treat the loss of bone minerals (no data).

L10 ANSWER 5 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:1329208 CAPLUS
DOCUMENT NUMBER: 146:161275
TITLE: HSV-1-mediated IL-1 receptor antagonist **gene therapy** ameliorates MOG35-55-induced experimental autoimmune encephalomyelitis in C57BL/6 mice
AUTHOR(S): Furlan, R.; Bergami, A.; Brambilla, E.; Butti, E.; De Simoni, M. G.; Campagnoli, M.; Marconi, P.; Comi, G.; Martino, G.
CORPORATE SOURCE: Neuroimmunology Unit, DIBIT, San Raffaele Scientific Institute, Milan, Italy
SOURCE: Gene Therapy (2007), 14(1), 93-98
CODEN: GETHEC; ISSN: 0969-7128
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Primary proinflammatory cytokines, such as IL-1β, play a crucial pathogenic role in multiple sclerosis and its animal model exptl. autoimmune encephalomyelitis (EAE), and may represent, therefore, a suitable therapeutic target. We have previously established the delivery of anti-inflammatory cytokine genes within the central nervous system (CNS), based on intracisternal (i.c.) injection of non-replicative HSV-1-derived vectors. Here we show the therapeutic efficacy of i.c. administration of an HSV-1-derived vector carrying the interleukin-1 receptor antagonist (IL-1ra) gene, the physiol. antagonist of the proinflammatory cytokine IL-1, in C57BL/6 mice affected by myelin oligodendrocyte glycoprotein-induced EAE. IL-1ra **gene therapy** is effective preventively, delaying EAE onset by almost 1 wk (22.4±1.4 days post-immunization vs 15.9±2.1 days in control mice; P=0.0229 log-**rank** test), and decreasing disease severity. Amelioration of EAE course was assocd. with a reduced no. of macrophages infiltrating the CNS and in a decreased level of proinflammatory cytokine mRNA in the CNS, suggesting an inhibitory activity of IL-1ra on effector cell recruitment, as **antigen**-specific peripheral T-cell activation and T-cell recruitment to the CNS is unaffected. Thus, local IL-1ra **gene therapy** may represent a therapeutic alternative for the inhibition of immune-mediated demyelination of the CNS.

OS.CITING REF COUNT: 16 THERE ARE 16 CAPLUS RECORDS THAT CITE THIS RECORD (16 CITINGS)

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 6 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Ref Referen ces

ACCESSION NUMBER: 2006:735981 CAPLUS

DOCUMENT NUMBER: 145:160139

TITLE: Methods of modifying CD11c+ dendritic cell development to form osteoclasts functional in the bone environment

INVENTOR(S): Teng, Yen-Tung A.

PATENT ASSIGNEE(S): University of Rochester, USA

SOURCE: PCT Int. Appl., 72 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006079051	A2	20060727	WO 2006-US2397	20060124
WO 2006079051	A3	20070201		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

US 20090028876	A1	20090129	US 2008-814515		20080416
PRIORITY APPLN. INFO.:			US 2005-646941P	P	20050124
			WO 2006-US2397	W	20060124

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB An ex vivo method of producing osteoclasts is described that includes providing isolated CD11c+ dendritic cells and culturing the CD11c+ dendritic cells in culture medium under conditions effective to produce osteoclasts. Also disclosed are methods of up-regulating or down-regulating bone resorption by manipulating the osteoclastogenesis of CD11c+ dendritic cells either in vivo or in vitro. Methods of treating an inflammatory bone disease or a metabolic bone disorder in a subject, and screening assays to identify compds. or genes that affect myeloid osteoclastogenesis are also described.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 7 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:201658 CAPLUS
DOCUMENT NUMBER: 145:186219
TITLE: Osteosarcoma: current status of immunotherapy and future trends (Review)
AUTHOR(S): Mori, Kanji; Redini, Francoise; Gouin, Francois; Cherrier, Bertrand; Heymann, Dominique
CORPORATE SOURCE: INSERM ERI 7, Physiopathologie de la Resorption Osseuse et Therapie des Tumeurs Osseuses Primitives, Faculte de Medecine, Universite de Nantes EA 3822, Nantes, 44035/1, Fr.
SOURCE: Oncology Reports (2006), 15(3), 693-700 CODEN: OCRPEW; ISSN: 1021-335X
PUBLISHER: Oncology Reports
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Osteosarcoma is the most common primary bone tumor and represents a major therapeutic challenge in medical oncol. While the use of aggressive chemotherapy has drastically improved the prognosis of the patients with nonmetastatic osteosarcomas, the very poor prognosis of patients with metastasis have led to the exploration of new, more effective and less toxic treatments, such as immunotherapy for curing osteosarcoma. Compared to the numerous reports describing successful immunotherapy for other solid tumors, the no. of reports concerning immunotherapy for osteosarcoma is low. However, this therapeutic strategy opens new areas for the treatment of osteosarcoma. In this review, the reasons for delay and all elements essential to develop immunotherapy concerning osteosarcoma are defined. Several pieces of evidence strongly support the potential capability of new therapies such as cellular therapy and **gene therapy** to eradicate osteosarcoma. Thus, clin. human trials using peptides, cytokines and dendritic cells have been performed. Tumor-infiltrating lymphocytes and some tumor **antigens** have been identified in osteosarcoma and resulted in an important breakthrough in cellular immunotherapy. Also, RANKL/**RANK**/OPG, the key regulator of bone metab., is a hot spot in this field as therapeutic tools. Immunotherapy for osteosarcomas has great potential, promising improvement in the survival rate and better quality of life for the patients.

OS.CITING REF COUNT: 10 THERE ARE 10 CAPLUS RECORDS THAT CITE THIS RECORD (10 CITINGS)
REFERENCE COUNT: 102 THERE ARE 102 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 8 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:395470 CAPLUS
DOCUMENT NUMBER: 142:442896
TITLE: Methods for differentiating stem cells using a self-replicating neocentromeric artificial chromosome with chromatin domains expressing transgenes for **gene therapy**
INVENTOR(S): Choo, Kong-Hong Andy; Wong, Lee Hwa; Saffery, Richard Eric
PATENT ASSIGNEE(S): Murdoch Childrens Research Institute, Australia
SOURCE: PCT Int. Appl., 168 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005040391	A1	20050506	WO 2004-AU1469	20041025

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: AU 2003-905894 A 20031027

AB The present invention relates to the field of tissue engineering and genetic manipulation of cells and to methods for generating tissue suitable for use in repair, replacement, rejuvenation or augmentation therapy. The present invention contemplates a method for genetically manipulating a stem cell by introducing a nucleic acid mol. comprising a centromere or neo-centromere into the stem cell, wherein the nucleic acid mol. conveys genetic information which is capable of introducing to or modifying a trait within the stem cell or progeny of the stem cell such as but not limited to modulating the level of stem cell proliferation, differentiation and/or self-renewal. The neo-centromere is devoid of α-satellite repeat DNA. One aspect of the present invention provides a stem cell comprising a self-replicating artificial chromosome with a neo-centromere having centromeric chromatin domains comprising expressible genetic material which modifies or introduces at least one trait in said stem cell. Microarray gene expression profiles were conducted for human 10q25 centromeric region. The engineered stem cells may also be re-programmed, for example, to direct the cells down a different cell lineage.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 9 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:413999 CAPLUS
DOCUMENT NUMBER: 139:2109
TITLE: cDNAs encoding human endokine α and their use in diagnosis and treatment of metabolic bone diseases
INVENTOR(S): Yu, Guo-Liang; Ni, Jian; Rosen, Craig A.; Nardelli, Bernardetta
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 145 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 20030100074	A1	20030529	US 2002-218547		20020815
US 7087225	B2	20060808			
WO 2003070763	A1	20030828	WO 2002-US25809		20020815
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2002366433	A1	20030909	AU 2002-366433		20020815
PRIORITY APPLN. INFO.:			US 2001-312542P	P	20010816
			US 2001-330761P	P	20011030
			WO 2002-US25809	W	20020815

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The present invention concerns methods for diagnosis and treatment of metabolic bone diseases and disorders using a novel member of the tumor necrosis factor (TNF) family of cytokines. In particular the invention provides methods of using the Endokine alpha protein and/or homomultimeric and/or heteromultimeric polypeptide complexes contg. Endokine alpha, in the diagnosis, prognosis and treatment of metabolic bone diseases and disorders. Also provided by the invention are methods of using the Endokine alpha protein and/or homomultimeric and/or heteromultimeric polypeptide complexes contg. Endokine alpha, in the diagnosis, prognosis and treatment of diseases and/or disorders assocd. with aberrant osteoclast development and/or activity. The present invention also provides isolated polynucleotides encoding polypeptides of the invention, antibodies thereto, and agonists and antagonists thereof, for use in the diagnosis, prognosis and treatment of metabolic bone diseases and disorders.

REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 10 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:966944 CAPLUS
DOCUMENT NUMBER: 138:37611
TITLE: **Gene therapy** approaches to HIV infection

AUTHOR(S): Lori, Franco; Guallini, Paola; Galluzzi, Luca; Lisziewicz, Julianna
CORPORATE SOURCE: Research Institute for Genetic and Human Therapy, IRCCS Policlinico S. Matteo, Pavia, Italy
SOURCE: American Journal of PharmacoGenomics (2002), 2(4), 245-252
CODEN: AJPMC8; ISSN: 1175-2203
PUBLISHER: Adis International Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. The HIV pandemic represents a new challenge to biomedical research. What began as a handful of recognized cases among homosexual men in the US has become a global pandemic of such proportions that it clearly **ranks** as one of the most destructive viral scourges in history. In the past few years new treatments and drugs have been developed and tested, but the development of a new generation of therapies remains a major priority, because of the lack of chemotherapeutic drugs or vaccines that show long-term efficacy in vivo. Recently, gene therapeutic strategies for the treatment of patients with HIV infection have received increased attention because they are able to offer the possibility of simultaneously targeting multiple sites in the HIV genome, thereby minimizing the prodn. of resistant virus. Recombinant genes for **gene therapy** can be classified as expressing interfering proteins (intracellular antibodies, dominant neg. proteins) or interfering RNAs (antisense RNAs, ribozymes, RNA decoys). The latter group offers the advantage of avoiding the stimulation of host immune response which might progressively decrease the efficacy of proteins. The stumbling block to achieving lasting antiviral effects is still represented by the lack of efficient gene transfer techniques capable of generating persistent transgene expression and a high no. of transduced cells relative to untransduced cells. Novel delivery vectors, such as lentiviruses, might overcome some of these shortcomings. The use of recombinant genes to generate immunity is a very promising concept that is rapidly expanding. Since the immune system can significantly amplify the response to tiny amts. of **antigen**, DNA vaccines can indeed be delivered by exploiting traditional **gene therapy** approaches without the need of high transduction efficiency.

OS.CITING REF COUNT: 12 THERE ARE 12 CAPLUS RECORDS THAT CITE THIS RECORD (12 CITINGS)
REFERENCE COUNT: 77 THERE ARE 77 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 11 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:228744 CAPLUS
DOCUMENT NUMBER: 134:247267
TITLE: Clostridial neurotoxin targeted conjugates for inhibition of secretion from non-neuronal cells
INVENTOR(S): Foster, Keith Alan; Chaddock, John Andrew; Purkiss, John Robert; Quinn, Conrad Padraig
PATENT ASSIGNEE(S): Microbiological Research Authority, UK
SOURCE: PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 2001021213	A2	20010329	WO 2000-GB3669	20000925
WO 2001021213	A3	20020711		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2383470	A1	20010329	CA 2000-2383470	20000925
EP 1235594	A2	20020904	EP 2000-962721	20000925
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003509476	T	20030311	JP 2001-524636	20000925
AU 782457	B2	20050728	AU 2000-74365	20000925
EP 2110142	A2	20091021	EP 2009-157032	20000925
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, AL, LT, LV, MK, RO, SI				
US 20030180289	A1	20030925	US 2002-88665	20020814
AU 2005227383	A1	20051124	AU 2005-227383	20051027
AU 2005227383	B2	20080821		
AU 2008241572	A1	20081127	AU 2008-241572	20081031
AU 2008241572	B2	20110127		

PRIORITY APPLN. INFO.:	GB 1999-22554	A	19990923
	EP 2000-962721	A3	20000925
	WO 2000-GB3669	W	20000925
	AU 2005-227383	A3	20051027

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB A method of treatment of disease by inhibition of cellular secretory processes is provided. The method has particular application in the treatment of diseases dependent on the exocytotic activity of endocrine cells, exocrine cells, inflammatory cells, cells of the immune system, cells of the cardiovascular system, and bone cells. Agents and compns. therefor, as well as methods for manufg. these agents and comps., are provided. In a preferred embodiment a clostridial neurotoxin, substantially devoid of holotoxin binding affinity for neuronal cells of the presynaptic muscular junction, is assocd. with a targeting moiety. The targeting moiety is selected such that the clostridial toxin conjugate so formed may be directed to a non-neuronal target cell to which the conjugate may bind. Following binding, a neurotoxin component of the conjugate, which is capable of inhibition of cellular secretion, passes into the cytosol of the target cell by cellular internalization mechanisms. Thereafter, inhibition of secretion from the target cell is effected.

OS.CITING REF COUNT:	5	THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)
REFERENCE COUNT:	8	THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 12 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER:	2000:452056 CAPLUS
DOCUMENT NUMBER:	134:98284
TITLE:	Isolation and characterization of CD34-low/negative mouse hematopoietic stem cells
AUTHOR(S):	Nakauchi, Hiromitsu; Osawa, Masatake; Sudo, Kazuhiro;

Ema, Hideo
CORPORATE SOURCE: Institute of Basic Medical Sciences and Center for TARA, University of Tsukuba, Tsukuba, 305-8575, Japan
SOURCE: Keio University Symposia for Life Science and Medicine (2000), 5(Cell Therapy), 95-103
CODEN: KUSMF9
PUBLISHER: Springer-Verlag Tokyo
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 16 refs. with an emphasis on the authors' research. We have previously reported that, in adult mouse bone marrow, CD34low/- c-Kit+ Sca-1+ lineage markers neg. (Lin-) (CD34-KSL) cells represent hematopoietic stem cells with long-term marrow repopulating ability whereas CD34+ c-Kit+ Sca-1+ Lin- (CD34+KSL) cells are progenitors with short-term reconstitution capacity. To characterize these two populations of cells further, relative expression of various genes was examd. by RT-PCR. In CD34-KSL cells, most cytokine receptor genes were not expressed with the exception of IL2Rγ and AIC-2B. In contrast, expression of all cytokine receptor genes examd. except IL-2Rα, IL-7Rα, and IL9Rα chains were found in CD34+KSL cells. Cell cycle studies revealed only 3% of CD34-KSL cells and 26% of CD34-KSL cells are dividing at a given time. Long-term BrdU administration study demonstrated, however, that majority of CD34-KSL cells contribute to hemopoiesis by dividing very slowly. Furthermore, anal. of aged mice revealed more than tenfold increase in abs. no. of CD34-KSL cells. Those CD34-KSL cells in aged mice appeared to include HPP-CFC at an equiv. frequency with those in younger mice. These data support our previous notion that CD34-KSL cells are at higher **rank** in hematopoietic hierarchy than CD34+KSL cells. In addn., our results provide important clues for cell therapy and **gene therapy** targeting hematopoietic stem cells.

REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 13 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2000:111338 CAPLUS
DOCUMENT NUMBER: 132:121345
TITLE: Treatment of established tumor is associated with ICAM-1 upregulation and reversed by CD8 depletion in a tumor necrosis factor-alpha gene transfected mouse mammary tumor
AUTHOR(S): Matory, Yvedt L.; Dorfman, David M.; Wu, Lei; Chen, Man; Goedegebuure, Peter; Eberlein, Timothy J.
CORPORATE SOURCE: Harvard Medical School, Brigham Women's Hospital, Boston, MA, 02115, USA
SOURCE: Pathobiology (2000), 67(4), 186-195
CODEN: PATHEF; ISSN: 1015-2008
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We have performed TNF-α gene transfection in a mouse mammary cancer line and found significant antitumor effects. We hypothesize that the antitumor effects obsd. in this model are mediated by ICAM-1 and by the recruitment of CD4+ and CD8+ T cells. In vivo (Balb/c mice) tumor growth inhibition, treatment of established tumor and the effects of ICAM-1 and CD4+ and CDS+ T cells were evaluated. Gene transfection with highly efficient vectors resulted in secretion of large amts. of TNF-α (ELISA). In vivo anti-tumor effects were tested. The no. of cells required to generate palpable tumor 7-10 days after s.c. injection was

detd. (1 × 106). The same no. of transfected cells were injected s.c. and compared to nontransfected controls. Tumors were measured blindly and size was analyzed on day 30 by the Wilcoxon **rank** sum test. Mean tumor size after injection of transfected cells is compared to that of controls. Control tumors reached the max. allowable size by day 30 (4 cm2). On day 30 EMT6-TNF-α tumors were 0.48 cm2. The effect of repeat injection was also tested. Animals were injected with transfected cells or wild-type control on day-6 and challenged with the same no. of wild-type tumor cells on day 0. Significant immune protection against subsequent challenge was seen after 1st time injection with EMT6-TNF-α but not after 1st time EMT6 wild-type injection (1.62 vs. 4 cm2). Treatment of 6-day-old tumor was also evaluated. On day 30, mean tumor size in animals treated with EMT6-TNF-α was 0.9 cm2 compared to 4 cm2 for controls. In all expts., CD8+ T cell depletion and CD4+ T cell depletion caused a reversal of TNF-α-induced inhibitory effects. In addn., in vivo antibody blocking of ICAM-1 in tumor growth expts. reversed antitumor effects (control 4 cm2, TNF-α 0.2 cm2, and ICAM-1 blocking 3.14 cm2). Using flow cytometry, MHC class I and II and ICAM-1 adhesion mol. expression of transfected tumor was tested. ICAM-I expression was significantly upregulated. MHC class II **antigen** expression was also increased. TNF-α-transfected human breast cancer was also evaluated. 3 Cell lines and fresh tumor were transfected to express TNF-α. In vitro anal. revealed ICAM-1 upregulation following transfection. Histol. anal. and immunohistochem. staining revealed TNF-α and ICAM-1 in transfected tumors and not in wild-type tumors. Highly significant in vivo tumor growth inhibition and immune protection after injection with TNF-α-transfected tumors appears to be mediated predominantly by CD8+ T cells and ICAM-1 upregulation. These findings suggest that TNF-α increases recruitment and adhesion of effector T cells.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 14 OF 18 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | Citing References

ACCESSION NUMBER: 2008:155958 BIOSIS
DOCUMENT NUMBER: PREV200800161746
TITLE: HSV-1-mediated IL-1 receptor antagonist **gene therapy** ameliorates MOG(35-55)-induced experimental autoimmune encephalomyelitis in C57BL/6 mice.
AUTHOR(S): Furlan, R. [Reprint Author]; Bergami, A.; Brambilla, E.; Butti, E.; De Simoni, M. G.; Campagnoli, M.; Marconi, P.; Comi, G.; Martino, G.
CORPORATE SOURCE: San Raffaele Sci Inst, Neuroimmunol Unit, DIBIT, Via Olgettina 58, I-20132 Milan, Italy
furlan.roberto@hsr.it
SOURCE: Gene Therapy, (JAN 2007) Vol. 14, No. 1, pp. 93-98. ISSN: 0969-7128.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Mar 2008
Last Updated on STN: 5 Mar 2008

AB Primary proinflammatory cytokines, such as IL-1 beta, play a crucial pathogenic role in multiple sclerosis and its animal model experimental autoimmune encephalomyelitis (EAE), and may represent, therefore, a suitable therapeutic target. We have previously established the delivery of anti-inflammatory cytokine genes within the central nervous system (CNS), based on intracisternal (i.c.) injection of non-replicative

HSV-1-derived vectors. Here we show the therapeutic efficacy of i.c. administration of an HSV-1-derived vector carrying the interleukin-1receptor antagonist (IL-1ra) gene, the physiological antagonist of the proinflammatory cytokine IL-1, in C57BL/6 mice affected by myelin oligodendrocyte glycoprotein-induced EAE. IL-1ra **gene therapy** is effective preventively, delaying EAE onset by almost 1 week (22.4 +/- 1.4 days post-immunization vs 15.9 +/- 2.1 days in control mice; P = 0.0229 log-**rank** test), and decreasing disease severity. Amelioration of EAE course was associated with a reduced number of macrophages infiltrating the CNS and in a decreased level of proinflammatory cytokine mRNA in the CNS, suggesting an inhibitory activity of IL-1ra on effector cell recruitment, as **antigen**-specific peripheral T-cell activation and T-cell recruitment to the CNS is unaffected. Thus, local IL-1ra **gene therapy** may represent a therapeutic alternative for the inhibition of immune-mediated demyelination of the CNS.

L10 ANSWER 15 OF 18 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on

Full Text | Claim References

STN

ACCESSION NUMBER: 2006:265332 BIOSIS
DOCUMENT NUMBER: PREV200600268984
TITLE: Osteosarcoma: Current status of immunotherapy and future trends (Review).
AUTHOR(S): Mori, Kanji [Reprint Author]; Redini, Francoise; Gouin, Frans; Cherrier, Bertrand; Heymann, Dominique
CORPORATE SOURCE: Univ Nantes, Fac Med, EA 3822, INSERM ERI 7, 1 Rue Gaston Veil, F-44035 Nantes 1, France
kanchi@belle.shiga-med.ac.jp
SOURCE: Oncology Reports, (MAR 2006) Vol. 15, No. 3, pp. 693-700. ISSN: 1021-335X.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 10 May 2006
Last Updated on STN: 10 May 2006

AB Osteosarcoma is the most common primary bone tumor and represents a major therapeutic challenge in medical oncology. While the use of aggressive chemotherapy has drastically improved the prognosis of the patients with non-metastatic osteosarcomas, the very poor prognosis of patients with metastasis have led to the exploration of new, more effective and less toxic treatments, such as immunotherapy for curing osteosarcoma. Compared to the numerous reports describing successful immunotherapy for other solid tumors, the number of reports concerning immunotherapy for osteosarcoma is low. However, this therapeutic strategy opens new areas for the treatment of osteosarcoma. In this review, the reasons for delay and all elements essential to develop immunotherapy concerning osteosarcoma are defined. Several pieces of evidence strongly support the potential capability of new therapies such as cellular therapy and **gene therapy** to eradicate osteosarcoma. Thus, clinical human trials using peptides, cytokines and dendritic cells have been performed. Tumor-infiltrating lymphocytes and some tumor **antigens** have been identified in osteosarcoma and resulted in an important breakthrough in cellular immunotherapy. Also, RANKL/**RANK**/OPG, the key regulator of bone metabolism, is a hot spot in this field as therapeutic tools. Immunotherapy for osteosarcomas has great potential, promising improvement in the survival rate and better quality of life for the patients.

L10 ANSWER 16 OF 18 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on

Full Text | String References

STN

ACCESSION NUMBER: 2002:459629 BIOSIS
DOCUMENT NUMBER: PREV200200459629
TITLE: Interleukin-12-gene transduction makes DCs from tumor-bearing mice an effective inducer of tumor-specific immunity in a peritoneal dissemination model.
AUTHOR(S): Furumoto, Katsuyoshi [Reprint author]; Mori, Akira; Yamasaki, Seiji; Inoue, Naoya; Yang, Weige; Nakau, Masayuki; Yasuda, Seiichi; Arii, Shigeki; Imamura, Masayuki
CORPORATE SOURCE: Department of Surgery and Surgical Basic Science, Graduate School of Medicine, Kyoto University, 54 Shogoin Kawara-cho, Sakyo-ku, Kyoto, 606-8507, Japan
furumoto@stanford.edu
SOURCE: Immunology Letters, (August 1, 2002) Vol. 83, No. 1, pp. 13-20. print.
CODEN: IMLED6. ISSN: 0165-2478.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Aug 2002
Last Updated on STN: 28 Aug 2002

AB Dendritic cells (DCs) from cancer patients, as well as tumor-infiltrating DCs, are reported to have suppressed immunostimulatory capacity. One of the major problems in the clinical use of DCs for treating tumors is that the DCs must be autologous ones obtained from patients. Compared with normal DCs (nDCs), flow-cytometric analysis and allogeneic mixed lymphocyte reaction (MLR) have revealed lower expression of the costimulatory molecules and suppressed T-cell-stimulatory activity in DCs derived from tumor-bearing mice (tDCs) despite of culture. We reported previously that the interleukin-12 (IL-12)-gene-transduced nDCs inhibited tumor growth due to induced tumor-specific Th1 and cytotoxic T cells (CTLs) in a murine established subcutaneous tumor model. In the present study, we examined whether tDCs could induce immune responses against tumors after IL-12-gene transduction in an established peritoneal dissemination model. The intraperitoneal injection of IL-12-gene-transduced tDCs resulted in prolonged survival of some treated mice (log-**rank** test; P = 0.001) and tumor-specific Th1 and CTL activity. The injection of IL-12-gene-transduced nDCs prolonged the survival of all treated mice (P < 0.0001) and elicited tumor-specific immunity, which were better than those of IL-12-gene-transduced tDCs. Taken together, DC modification of IL-12-gene transduction is an effective and promising approach for cancer therapy even when immunosuppressive tDCs are employed.

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Full Text | String References

STN

ACCESSION NUMBER: 2000:256574 BIOSIS
DOCUMENT NUMBER: PREV200000256574
TITLE: The liver as a life-guard.
AUTHOR(S): Ramadori, Giuliano [Reprint author]; Armbrust, Thomas
CORPORATE SOURCE: Center of Internal Medicine, Department of Gastroenteroloy and Endocrinology, Georg-August-University, Robert-Koch-Strasse 40, 37075, Goettingen, Germany
SOURCE: Giornale Italiano di Malattie Infettive, (July-Aug., 1999) Vol. 5, No. 4, pp. 209-216. print.
ISSN: 1126-9952.
DOCUMENT TYPE: Article
General Review; (Literature Review)

LANGUAGE: English
ENTRY DATE: Entered STN: 21 Jun 2000
Last Updated on STN: 5 Jan 2002

AB Clearance of endogenous or foreign, soluble or particulate matter may **rank** as the most important 'every day' defense strategy of the liver involving as many as three different cell populations within that organ (KC, EC, HC). With the gut in the back the presence of the, by far, largest population of resident tissue macrophages indicates the need for a strong and efficient clearance of foreign material preventing their entry into systemic circulation. The capacity of KC to release a broad spectrum of powerful molecules in the state of activation may **rank** as part of this function since endocytosis is the main mechanism of KC activation. Beneath clearance, the liver is providing much more that seems to be essential in defense. The acute phase response, the systemic alterations seen in infection, tissue damage or other inflammatory reactions, to a major extend, can be induced, mediated or executed by the liver. Although not completely understood the acute phase response is suggested to ease the resolvement of those pathological states. Another constitutive action of the liver is the oral tolerance, the suppression of the immune response to portal **antigens**. It is likely that this phenomenon mediated by active suppression is essential in preventing hyperresponsiveness to foreign material (food components) and endogenous molecules shed from gut cells and reaching the blood stream. Oral tolerance seems to be suited to enable development of new strategies in fighting diseases. It gained new actuality in **gene therapy**. Gene delivery by adenoviruses is limited by a strong immune response against adenoviral **antigens**, but oral administration of adenoviral **antigens** can sustain efficient expression of the transferred genes.

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Full Text | Cited References

ACCESSION NUMBER: 2000:133734 BIOSIS
DOCUMENT NUMBER: PREV200000133734
TITLE: Treatment of established tumor is associated with ICAM-1 upregulation and reversed by CD8 depletion in a tumor necrosis factor-alpha gene transfected mouse mammary tumor.
AUTHOR(S): Matory, Yvedt L. [Reprint author]; Dorfman, David M.; Wu, Lei; Chen, Man; Goedegebuure, Peter; Eberlein, Timothy J.
CORPORATE SOURCE: Brigham and Women's Hospital, 75 Francis Street, Boston, MA, 02115, USA
SOURCE: Pathobiology, (July-Aug., 1999) Vol. 67, No. 4, pp. 186-195. print.
CODEN: PATHEF. ISSN: 1015-2008.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Apr 2000
Last Updated on STN: 4 Jan 2002

AB Introduction: We have performed TNF-alpha gene transfection in a mouse mammary cancer line and found significant antitumor effects. We hypothesize that the antitumor effects observed in this model are mediated by ICAM-1 and by the recruitment of CD4+ and CD8+ T cells. In vivo (Balb/c mice) tumor growth inhibition, treatment of established tumor and the effects of ICAM-1 and CD4+ and CD8+ T cells were evaluated. Methods and Results: Gene transfection with highly efficient vectors resulted in secretion of large amounts of TNF-alpha (ELISA). In vivo anti-tumor effects were tested. The number of cells required to generate palpable tumor 7-10 days after subcutaneous injection was determined (1 X 106). The same number of transfected cells were injected subcutaneously and

compared to nontransfected controls. Tumors were measured blindly and size was analyzed on day 30 by the Wilcoxon **rank** sum test. Mean tumor size after injection of transfected cells is compared to that of controls. Control tumors reached the maximum allowable size by day 30 (4 cm2). On day 30 EMT6-TNF-alpha tumors were 0.48 cm2 ($p < 0.05$). The effect of repeat injection (challenge was also tested. Animals were injected with transfected cells or wild-type control on day-6 and challenged with the same number of wild-type tumor cells on day 0. Significant immune protection against subsequent challenge was seen after first time injection with EMT6-TNF-alpha but not after first time EMT6 wild-type injection (1.62 vs. 4 cm2). Treatment of 6-day-old tumor was also evaluated. On day 30, mean tumor size in animals treated with EMT6-TNF-alpha was 0.9 cm2 compared to 4 cm2 for controls. In all experiments, CD8+ T cell depletion and CD4+ T cell depletion caused a reversal of TNF-alpha-induced inhibitory effects. In addition, in vivo antibody blocking of ICAM-1 in tumor growth experiments reversed antitumor effects (control 4 cm2, TNF-alpha 0.2 cm2 and ICAM-1 blocking 3.14 cm2). Using flow cytometry, MHC class I and II and ICAM-1 adhesion molecule expression of transfected tumor was tested. ICAM-1 expression was significantly upregulated. MHC class II **antigen** expression was also increased. TNF-alpha-transfected human breast cancer was also evaluated. Three cell lines and fresh tumor were transfected to express TNF-alpha. In vitro analysis revealed ICAM-1 upregulation following transfection. Histologic analysis and immunohistochemical staining revealed TNF-alpha and ICAM-1 in transfected tumors and not in wild-type tumors. Conclusion: Highly significant in vivo tumor growth inhibition and immune protection after injection with TNF-alpha-transfected tumors appears to be mediated predominantly by CD8+ T cells and ICAM-1 upregulation. These findings suggest that TNF-alpha increases recruitment and adhesion of effector T cells.

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=> Jun (s) Ap-1)
UNMATCHED RIGHT PARENTHESIS 'AP-1)'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> file caplus biosis
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                144.64     144.87

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)      SINCE FILE      TOTAL
                                                   ENTRY    SESSION
CA SUBSCRIBER PRICE                                -17.40     -17.40

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=> (c-jun) (s) (Ap-1)
L11        5357 (C-JUN) (S) (AP-1)

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L12          8 L11 AND RANK

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L12  ANSWER 1 OF 8  CAPLUS  COPYRIGHT 2011 ACS on STN
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Full Text | Citing References

ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004112706	A2	20041229	WO 2004-US19028	20040614
WO 2004112706	A3	20050414		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004249191	A1	20041229	AU 2004-249191	20040614
AU 2004249191	B2	20110106		
CA 2529051	A1	20041229	CA 2004-2529051	20040614
EP 1633372	A2	20060315	EP 2004-755303	20040614
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
JP 2007502868	T	20070215	JP 2006-533794	20040614
US 20070104686	A1	20070510	US 2004-560653	20040614

PRIORITY APPLN. INFO.:		
US 2003-478187P	P	20030613
US 2003-478230P	P	20030613
US 2003-478250P	P	20030613
WO 2004-US19028	W	20040614

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, **c-jun**, Sp-1, **AP-1**, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, **RANK**, **RANK** ligand, Ox40, Ox40 ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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Full Text | Citing References

ACCESSION NUMBER: 2004:332163 CAPLUS
DOCUMENT NUMBER: 140:404155
TITLE: Extracellular ATP activates c-jun N-terminal kinase signaling and cell cycle progression in hepatocytes
AUTHOR(S): Thevananther, Sundararajah; Sun, Hongdan; Li, Duo; Arjunan, Vijaya; Awad, Samir S.; Wyllie, Samuel; Zimmerman, Tracy L.; Goss, John A.; Karpen, Saul J.
CORPORATE SOURCE: Department of Pediatrics, Section of Gastroenterology, Hepatology and Nutrition, Baylor College of Medicine, Houston, TX, USA
SOURCE: Hepatology (Hoboken, NJ, United States) (2004), 39(2), 393-402
CODEN: HPTLD9; ISSN: 0270-9139
PUBLISHER: John Wiley & Sons, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Partial hepatectomy leads to an orchestrated regenerative response, activating a cascade of cell signaling events necessary for cell cycle progression and proliferation of hepatocytes. However, the identity of the humoral factors that trigger the activation of these pathways in the concerted regenerative response in hepatocytes remains elusive. In recent years, extracellular ATP has emerged as a rapidly acting signaling mol. that influences a variety of liver functions, but its role in hepatocyte growth and regeneration is unknown. In this study, we sought to det. if purinergic signaling can lead to the activation of c-jun N-terminal kinase (JNK), a known central player in hepatocyte proliferation and liver regeneration. Hepatocyte treatment with ATPγS, a nonhydrolyzable ATP analog, recapitulated early signaling events assocd. with liver regeneration-i.e., rapid and transient activation of JNK signaling, induction of immediate early genes c-fos and **c-jun**, and activator protein-1 (**AP-1**) DNA-binding activity. The **rank** order of agonist preference, UTP>ATP>ATPγS, suggests that the effects of extracellular ATP is mediated through the activation of P2Y2 receptors in hepatocytes. ATPγS treatment alone and in combination with epidermal growth factor (EGF) substantially increased cyclin D1 and proliferating cell nuclear antigen (PCNA) protein expression and hepatocyte proliferation in vitro. Extracellular ATP as low as 10 nM was sufficient to potentiate EGF-induced cyclin D1 expression. Infusion of ATP by way of the portal vein directly activated hepatic JNK signaling, while infusion of a P2 purinergic receptor antagonist prior to partial hepatectomy inhibited JNK activation. In conclusion, extracellular ATP is a hepatic mitogen that can activate JNK signaling and hepatocyte proliferation in vitro and initiate JNK signaling in regenerating liver in vivo. These findings have implications for enhancing our understanding of novel factors involved in the initiation of regeneration, liver growth, and development.

OS.CITING REF COUNT: 42 THERE ARE 42 CAPLUS RECORDS THAT CITE THIS RECORD (42 CITINGS)
REFERENCE COUNT: 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:619468 CAPLUS
DOCUMENT NUMBER: 137:349704
TITLE: TAK1-dependent activation of **AP-1** and **c-Jun** N-terminal kinase by receptor activator of NF-κB
AUTHOR(S): Lee, Soo Woong; Han, Sang-In; Kim, Hong-Hee; Lee, Zang Hee
CORPORATE SOURCE: Research Center for Proteineous Materials, School of Dentistry, Chosun University, GWangju, S. Korea
SOURCE: Journal of Biochemistry and Molecular Biology (2002), 35(4), 371-376
CODEN: JBMBE5; ISSN: 1225-8687
PUBLISHER: Springer-Verlag Singapore Pte. Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The receptor activator of nuclear factor kappa B (**RANK**) is a member of the tumor necrosis factor (TNF) receptor superfamily. It plays a crit. role in osteoclast differentiation, lymph node organogenesis, and mammary gland development. The stimulation of **RANK** causes the activation of transcription factors NF-κB and activator protein 1 (AP1), and the mitogen activated protein kinase (MAPK) c-Jun N-terminal kinase (JNK). In the signal transduction of **RANK**, the recruitment of the adaptor mols., TNF receptor-assocd. factors (TRAFs), is an initial cytoplasmic event. Recently, the assocn. of the MAPK kinase kinase, transforming growth factor-β-activated kinase 1 (TAK1), with TRAF6 was shown to mediate the IL-1 signaling to NF-κB and JNK. We investigated whether or not TAK1 plays a role in **RANK** signaling. A dominant-neg. form of TAK1 was discovered to abolish the **RANK**-induced activation of AP1 and JNK. The AP1 activation by TRAF2, TRAF5, and TRAF6 was also greatly suppressed by the dominant-neg. TAK1. The inhibitory effect of the TAK1 mutant on **RANK**- and TRAF-induced NF-κB activation was also obsd., but less efficiently. Our findings indicate that TAK1 is involved in the MAPK cascade and NF-κB pathway that is activated by **RANK**.

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Full Text | Citing References

ACCESSION NUMBER: 2000:893932 CAPLUS
DOCUMENT NUMBER: 134:158047
TITLE: Activation of c-Jun N-terminal kinase and activator protein 1 by receptor activator of nuclear factor κB
AUTHOR(S): Lee, Zang Hee; Kwack, Kyubum; Kim, Kyung Keun; Lee, Sang Ho; Kim, Hong-Hee
CORPORATE SOURCE: Department of Microbiology and Immunology, Chosun University Dental School, Kwangju, S. Korea
SOURCE: Molecular Pharmacology (2000), 58(6), 1536-1545
CODEN: MOPMA3; ISSN: 0026-895X
PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Receptor activator of nuclear factor κB (**RANK**), a lately identified member of the tumor necrosis factor receptor superfamily, plays important roles both in osteoclastogenesis and in lymph node development.

Previously, the authors and others showed that **RANK** could stimulate the activity of c-Jun N-terminal kinase (JNK). In this study, the authors investigated the mechanism by which **RANK** activates JNK. The authors found that N-terminal deletion mutants of tumor necrosis factor receptor-assocd. factor 2 and 6 were inhibitory to **RANK** activation of JNK. The JNK activation by **RANK** was also reduced by contransfection of kinase-inactive mutants of apoptosis signal-regulating kinase 1, MAPK/ERK kinase kinase 1, and nuclear factor κB-inducing kinase. In addn., dominant neg. mutants of Rac and Ras decreased the **RANK** stimulation of JNK activity. Furthermore, the authors detd. whether the **RANK** engagement of JNK signaling pathways could lead to the activation of the activator protein 1 (AP-1) transcription factor, one of the potential downstream targets of activated JNK. **RANK** was found to activate AP-1 in a manner dependent on the signaling mols. involved in the JNK activation by this receptor. Furthermore, the activation of JNK and ERK, but not that of p38, appeared to be involved in the AP-1 activation by **RANK**. Thus, **RANK** may use both JNK and ERK pathways to signal to the AP-1 transcription factor.

OS.CITING REF COUNT: 38 THERE ARE 38 CAPLUS RECORDS THAT CITE THIS RECORD (38 CITINGS)

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2000:494471 CAPLUS
DOCUMENT NUMBER: 133:160074
TITLE: Estrogens suppress **RANK** ligand-induced osteoclast differentiation via a stromal cell independent mechanism involving c-Jun repression
AUTHOR(S): Shevde, Nirupama K.; Bendixen, Amy C.; Dienger, Krista M.; Pike, J. Wesley
CORPORATE SOURCE: Department of Molecular and Cellular Physiology, University of Cincinnati, Cincinnati, OH, 45267, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2000), 97(14), 7829-7834
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Loss of ovarian function following menopause results in a substantial increase in bone turnover and a crit. imbalance between bone formation and resorption. This imbalance leads to a progressive loss of trabecular bone mass and eventually osteoporosis, in part the result of increased osteoclastogenesis. Enhanced formation of functional osteoclasts appears to be the result of increased elaboration by support cells of osteoclastogenic cytokines such as IL-1, tumor necrosis factor, and IL-6, all of which are neg. regulated by estrogens. The authors show here that estrogen can suppress receptor activator of NF-κB ligand (RANKL) and macrophage colony-stimulating factor (M-CSF)-induced differentiation of myelomonocytic precursors into multinucleated tartrate-resistant acid phosphatase-pos. osteoclasts through an estrogen receptor-dependent mechanism that does not require mediation by stromal cells. This suppression is dose-dependent, isomer-specific, and reversed by ICI 182780. Furthermore, the bone-sparing analogs tamoxifen and raloxifene mimic estrogen's effects. Estrogen blocks RANKL/M-CSF-induced activator protein-1-dependent transcription, likely through direct regulation of c-Jun activity. This effect is the result of a classical nuclear activity by estrogen receptor to regulate both c-Jun expression and its

phosphorylation by c-Jun N-terminal kinase. The authors' results suggest that estrogen modulates osteoclast formation both by down-regulating the expression of osteoclastogenic cytokines from supportive cells and by directly suppressing RANKL-induced osteoclast differentiation.

OS.CITING REF COUNT: 195 THERE ARE 195 CAPLUS RECORDS THAT CITE THIS RECORD (195 CITINGS)

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Chng References

ACCESSION NUMBER: 2000:114343 CAPLUS
DOCUMENT NUMBER: 132:234856
TITLE: Fosl1 is a transcriptional target of c-Fos during osteoclast differentiation
AUTHOR(S): Matsuo, Koichi; Owens, Jane M.; Tonko, Martin; Elliott, Candace; Chambers, Timothy J.; Wagner, Erwin F.
CORPORATE SOURCE: Research Institute of Molecular Pathology, Vienna, Austria
SOURCE: Nature Genetics (2000), 24(2), 184-187
CODEN: NGENEC; ISSN: 1061-4036
PUBLISHER: Nature America
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Osteoclasts are bone-resorbing cells derived from hematopoietic precursors of the monocyte-macrophage lineage. Mice lacking Fos (encoding c-Fos) develop osteopetrosis due to an early differentiation block in the osteoclast lineage1-3, c-Fos is a component of the dimeric transcription factor activator protein-1 (**Ap-1**), which is composed mainly of Fos (c-Fos, FosB, Fra-1 and Fra-2) and Jun proteins (**c-Jun**, JunB and JunD). Unlike Fra-1 (encoded by Fosl1), c-Fos contains transactivation domains required for oncogenesis and cellular transformation. The mechanism by which c-Fos exerts its specific function in osteoclast differentiation is not understood. Here we show by retroviral-gene transfer that all four Fos proteins, but not the Jun proteins, rescue the differentiation block in vitro. Structure-function anal. demonstrated that the major carboxy-terminal transactivation domains of c-Fos and FosB are dispensable and that Fra-1 (which lacks transactivation domains) has the highest rescue activity. Moreover, a transgene expressing Fra-1 rescues the osteopetrosis of c-Fos-mutant mice in vivo. The osteoclast differentiation factor RankI (also known as TRANCE, ODF and OPGL) induces transcription of Fosl1 in a c-Fos-dependent manner, thereby establishing a link between **Rank** signaling and the expression of Ap-1 proteins in osteoclast differentiation.

OS.CITING REF COUNT: 154 THERE ARE 154 CAPLUS RECORDS THAT CITE THIS RECORD (154 CITINGS)

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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Full Text | Chng References

ACCESSION NUMBER: 2004:191758 BIOSIS
DOCUMENT NUMBER: PREV200400180228
TITLE: Extracellular ATP activates c-jun N-terminal kinase signaling and cell cycle progression in hepatocytes.
AUTHOR(S): Thevananther, Sundararajah [Reprint Author]; Sun, Hongdan; Li, Duo; Arjunan, Vijaya; Awad, Samir S.; Wyllie, Samuel;

Zimmerman, Tracy L.; Goss, John A.; Karpen, Saul J.
CORPORATE SOURCE: Department of Pediatrics, Section of Gastroenterology, Hepatology and Nutrition, Texas Children's Liver Center, Baylor College of Medicine, One Baylor Plaza, MC 3-3391, Houston, TX, 77030, USA
sundarat@bcm.tmc.edu
SOURCE: Hepatology, (February 2004) Vol. 39, No. 2, pp. 393-402. print.
ISSN: 0270-9139 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Apr 2004
Last Updated on STN: 7 Apr 2004

AB Partial hepatectomy leads to an orchestrated regenerative response, activating a cascade of cell signaling events necessary for cell cycle progression and proliferation of hepatocytes. However, the identity of the humoral factors that trigger the activation of these pathways in the concerted regenerative response in hepatocytes remains elusive. In recent years, extracellular ATP has emerged as a rapidly acting signaling molecule that influences a variety of liver functions, but its role in hepatocyte growth and regeneration is unknown. In this study, we sought to determine if purinergic signaling can lead to the activation of c-jun N-terminal kinase (JNK), a known central player in hepatocyte proliferation and liver regeneration. Hepatocyte treatment with ATPgammaS, a nonhydrolyzable ATP analog, recapitulated early signaling events associated with liver regeneration-that is, rapid and transient activation of JNK signaling, induction of immediate early genes c-fos and **c-jun**, and activator protein-1 (**AP-1**) DNA-binding activity. The **rank** order of agonist preference, UTP>ATP>ATPgammaS, suggests that the effects of extracellular ATP is mediated through the activation of P2Y2 receptors in hepatocytes. ATPgammaS treatment alone and in combination with epidermal growth factor (EGF) substantially increased cyclin D1 and proliferating cell nuclear antigen (PCNA) protein expression and hepatocyte proliferation in vitro. Extracellular ATP as low as 10 nM was sufficient to potentiate EGF-induced cyclin D1 expression. Infusion of ATP by way of the portal vein directly activated hepatic JNK signaling, while infusion of a P2 purinergic receptor antagonist prior to partial hepatectomy inhibited JNK activation. In conclusion, extracellular ATP is a hepatic mitogen that can activate JNK signaling and hepatocyte proliferation in vitro and initiate JNK signaling in regenerating liver in vivo. These findings have implications for enhancing our understanding of novel factors involved in the initiation of regeneration, liver growth, and development.

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ACCESSION NUMBER: 2000:178964 BIOSIS
DOCUMENT NUMBER: PREV200000178964
TITLE: Fosl1 is a transcriptional target of c-Fos during osteoclast differentiation.
AUTHOR(S): Matsuo, Koichi; Owens, Jane M.; Tonko, Martin; Elliott, Candace; Chambers, Timothy J.; Wagner, Erwin F. [Reprint author]
CORPORATE SOURCE: Research Institute of Molecular Pathology, Vienna, Austria
SOURCE: Nature Genetics, (Feb., 2000) Vol. 24, No. 2, pp. 184-187. print.
ISSN: 1061-4036.
DOCUMENT TYPE: Article
LANGUAGE: English

ENTRY DATE: Entered STN: 11 May 2000
Last Updated on STN: 4 Jan 2002

AB Osteoclasts are bone-resorbing cells derived from haematopoietic precursors of the monocyte-macrophage lineage. Mice lacking Fos (encoding c-Fos) develop osteopetrosis due to an early differentiation block in the osteoclast lineage. c-Fos is a component of the dimeric transcription factor activator protein-1 (**Ap-1**), which is composed mainly of Fos (c-Fos, FosB, Fra-1 and Fra-2) and Jun proteins (**c-Jun**, JunB and JunD). Unlike Fra-1 (encoded by Fosl1), c-Fos contains transactivation domains required for oncogenesis and cellular transformation. The mechanism by which c-Fos exerts its specific function in osteoclast differentiation is not understood. Here we show by retroviral-gene transfer that all four Fos proteins, but not the Jun proteins, rescue the differentiation block in vitro. Structure-function analysis demonstrated that the major carboxy-terminal transactivation domains of c-Fos and FosB are dispensable and that Fra-1 (which lacks transactivation domains) has the highest rescue activity. Moreover, a transgene expressing Fra-1 rescues the osteopetrosis of c-Fos-mutant mice in vivo. The osteoclast differentiation factor Rankl (also known as TRANCE, ODF and OPGL; refs 8-11) induces transcription of Fosl1 in a c-Fos-dependent manner, thereby establishing a link between **Rank** signalling and the expression of Ap-1 proteins in osteoclast differentiation.

=>